EXHIBIT F

EXPERT REPORT OF EUGENE J. MARK, MD

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EXPERT REPORT OF EUGENE J. MARK, M.D.

I have been retained by the law firm of Dies & Hile to provide expert testimony concerning the potential health effects of exposure to asbestos including non-occupational and/or environmental exposures, such as that which could occur in buildings which contain friable asbestos-containing material. I may offer testimony concerning the disease processes with regard to the health effects of exposure to asbestos including diffuse malignant mesothelioma, carcinoma of the lung, parenchymal asbestosis, and pleural hyaline plaque. I may discuss the pathology of such asbestos-related diseases and the carcinogenic processes to the malignancies.

I am a medical doctor and my qualifications and experience are set forth in my attached CV.

In addition to asbestosis and lung cancer, exposure to asbestos fibers can induce diffuse malignant mesothelioma. Diffuse malignant mesothelioma can be caused by low exposures to asbestos and/or for brief periods of time. It is my opinion that exposure to asbestos is the cause of virtually all cases of diffuse malignant mesothelioma in the United States and that there is virtually no background incidence for this disease apart from exposure to asbestos (e.g., 1,2). Genetic susceptibility can play a role in the development of the tumor.

It is my opinion that there is no known safe level of exposure to asbestos. Over a broad range, the amount of asbestos inhaled correlates with the risk of developing most asbestos-related diseases. All types of asbestos can cause lung cancer and mesothelioma. It is my opinion that cancer and asbestosis are independent processes and that a patient need not have asbestosis in order to attribute a lung cancer to asbestos exposure.

There is substantial evidence in the medical and scientific literature that non-occupational, environmental or bystander exposure to asbestos at low levels and/or for a brief period of time is capable of causing the disease of mesothelioma (e.g. 3,4). Thus, there is concern for building occupants who may be exposed to friable and/or damaged asbestos-containing materials.

I may testify concerning the carcinogenicity of asbestos which becomes airborne and inhaled from asbestos--containing materials and the hazards associated with exposure to workers or bystanders or household members (e.g., 5-7). Any special exposure, that is, an exposure where there is scientific evidence to suggest induction of disease based on medicine, pathology, epidemiology, molecular pathology, physics, occupational health, medical history, toxicology and experimental studies, is damaging at the cellular level and therefore potentially damaging at the clinical level. Exposures to asbestos are considered to be cumulative in that each and every

exposure will contribute to the development of carcinoma and diffuse malignant mesothelioma in a patient who develops these diseases. In my opinion, persons who would come in contact with friable asbestos-containing materials in buildings which release asbestos fibers would have an increase in their risk of developing most asbestos-related diseases including carcinoma and diffuse malignant mesothelioma.

In my clinical practice, I have reviewed records and pathology from individuals with exposure to asbestos in place in buildings, and I have concluded in such instances that such exposure to and inhalation of asbestos caused or contributed to cause asbestos-related diseases including asbestosis, pleural hyaline plaque, carcinoma of the lung, and diffuse malignant mesothelioma. Exposure to and inhalation from asbestos-containing materials which becomes airborne and enters the breathing zone can cause asbestos-related diseases.[1]

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2 Welch LS, Acherman YIZ, Haile E, et al: Asbestos and peritoneal mesothelioma among college-educated men. Int J Occup Environ Health 2005; 11:254-258.

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4 Mark EJ, Kradin RL: Pathological recognition of diffuse malignant mesothelioma of the pleura: the significance of the historical perspective as regards this signal tumor. Sem Diagn Pathol 2006; 23:25-34.

5. Oliver, L.C., et al., 1991, Disease Resulting from Asbestos Exposure in Buildings, Third Wave of Asbestos Disease: Exposure to Asbestos in Place, Annals of NY Acad of Sciences, Volume 643; Levin, S.M., et al.,

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7. Anderson, H.A., et al., Mesothelioma Among Employees with Likely Contact with In-Place Asbestos-Containing Building Materials, Third Wave of Asbestos Disease: Exposure to Asbestos in Place, Annals of NY Acad of Sciences, Volume 643.

Eugene J. Mark MD

Eugene J. Mark, M.D.

11 Jan 2007

Date

I may further testify consistent with my testimony in my deposition in *State of Hawaii v. W.R. Grace & Co.-Conn., et al.*, No. 93-4161-10, in the Circuit Court of the First Circuit, State of Hawaii.

Eugene J. Mark MD

Eugene J. Mark, M.D.

11 Jan 2007

Date

Seminars in Diagnostic Pathology (2006) 23, 25-34

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Seminars in Diagnostic Pathology

Pathological recognition of diffuse malignant mesothelioma of the pleura: the significance of the historical perspective as regards this signal tumor

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KEYWORDS
Diffuse malignant mesothelioma;
Asbestos;
History

Diffuse malignant mesothelioma (DMM) is a distinctive tumor which provides an uncommon opportunity to observe the gradual appreciation and increasing incidence of a new disease. DMM is a new disease. One cannot comment intelligently about the pathology of sporadic cases that might have occurred before the beginnings of anatomic pathology, but we do know that there were so few cases before 1930 that the very existence of the disease was not accepted in general before 1930 and not accepted by all pathologists even up until 1960. Because DMM is increasing on a worldwide basis and is making its appearance in the developing world, where it has not previously been diagnosed, appreciation of how the disease came to be noticed sheds light on its causation. As a signal tumor for exposure to asbestos, and knowing that all special exposures contribute to the development of the disease, knowledge of its continuing escalation underscores the importance of recognition of previously unimplicated or occult exposures for reasons of public health in both developed and developing countries.
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Because diffuse malignant mesothelioma (DMM) is virtually only caused by exposure to asbestos,[1-3] because it is a signal tumor for exposure to asbestos,[4] because all commercial forms of asbestos are known to be human carcinogens,[5] because there is no threshold level of exposure to asbestos below which there is no risk of DMM,[6] because all special exposures to asbestos together contribute to cause DMM based on modern understanding of cellular and molecular pathology in the multistage pathway of oncogenesis[7] and calculation of mortality rates based on cumulative lifetime exposure,[3] because DMM is almost universally fatal,[8] and because DMM has reached epidemic proportions in certain populations,[9] efforts at understanding how it came to be recognized and why it is a signal tumor for as yet unappreciated exposures to asbestos seem warranted.

This paper begins with a background of some historical aspects of asbestos in general and then focuses on those pertaining to DMM. It traces the timing of the literature and singles out key moments in the description of the disease. The importance of the timing of recognition of DMM lies in the information that it provides on the histogenesis of the tumor. Reports in the literature vary as to what proportion of persons who develop DMM have been exposed to asbestos. The frequency with which a positive history of exposure emerges depends on how and by whom the history is obtained, including the training and experience of the interviewer.[10,11] It also depends on whether the history is recorded in the medical records or elsewhere and on what constitutes a positive history.

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This paper discusses DMM of the pleura in humans. It does not cover experimentally induced mesotheliomas in animals. It does not cover the separate condition of mesotheliomas in infants and children. It does not cover DMM in the peritoneum or the pericardium, except to indicate that DMM is the same serosal tumor whether in begins in the pleura or the peritoneum or the pericardium based on basic principles of biology. The link of DMM of the peritoneum to asbestos[12,13] parallels that of DMM of the pleura. Much of the reported difference in the two sites of disease can be accounted for by the different specialty journals in which these two conditions generally are reported.

The documents to study the history of asbestos-related lung disease

The sources for understanding the appearance of DMM and other asbestos-related diseases rely on various sources. General medical literature, dating back to the early 1900s on asbestos bodies and asbestosis, provides first descriptions. Specialty medical journals expand on variations of the diseases and their diagnosis and treatment. Governmental documents, dating back to 1930 or earlier, itemize requirements on industry for public health. Industry documents describe what is known at a given time by manufacturers or distributors. Organized labor disseminated information about health concerns and protective measures by trade journals and leaflets. Labels on products synopsize the potential dangers inherent in the use of the product.

The geology of asbestos

The word asbestos is said to be derived from the Greek "unquenchable," as in the unquenchable wick for the perpetual flame to the Goddess Athene. Webster's dictionary may define it as a grayish or greenish variety of amphibole occurring in long delicate fibers or in fibrous masses. A geologist may think of asbestos as a certain type of rock in a certain location. A minerologist may define it as certain families of fibrous minerals with crystalline arrays. A chemist may define its composition as a ribbon of silica with hydroxyl groups and magnesium, calcium, iron, and sodium anions. Electron microscopists think of the shape of individual fibers.

The meaning of asbestos can depend on one's vocation and avocation. Builders and tradesmen and businesses think of its commercial uses. An industrial engineer may itemize its properties for resistance to fire and heat and relative insolubility in strong acid and alkali. Physicians think of its diseases. Biologists use it to experimentally induce inflammation or malignancy. Judges think of how it has occupied the courts. Legislators think of the costs to recover damages caused by the substance.

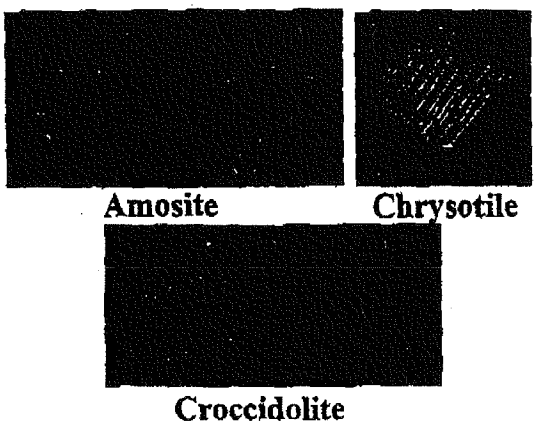

Figure 1 Asbestos in its natural state in rock. The three major forms appear as lighter colored layers in their vaunted colors of brown (amosite), white (chrysotile), and blue (crocidolite).

Categories of asbestos

Asbestos is generally divided into two major groups: amphibole asbestos and serpentine asbestos. The common amphiboles are crocidolite and amosite but also include tremolite, actinolite, anthophyllite, and others. All of the amphiboles can be described chemically as hydrated magnesium silicates but differ in their elemental content and ionic structure. The common serpentine is chrysotile.

Crocidolite (blue asbestos) in nature is a pale light blue, amosite (brown asbestos) tends to dirty brown, and chrysotile (white asbestos) is light in color when compared with crocidolite and amosite. The appearance of the minerals in their natural state is illustrated in Figure 1. All of the forms of asbestos cause DMM, but there are differences in the fibrogenicity and oncogenicity, with crocidolite being the most oncogenic of the three and chrysotile the least.

In addition to asbestos, fibrous minerals with similar chemical composition and described as asbestiform also can cause disease. These include principally erionite and balangerite, and these asbestiform fibers also cause diffuse malignant mesothelioma. Erionite, a fibrous zeolite found principally in the Capadocchia region of Anatolia, Turkey (Figure 2), as well as in other areas of the earth, has caused endemic DMM. The asbestiform minerals and radiotherapy are the only established cause of DMM apart from asbestos, a fact which further highlights the unique role of asbestos in causation of DMM.

Ancient usage of asbestos

Asbestos has been found incorporated into Finish pottery dating from approximately 2500 BC. In ancient Greece (approximately 500 BC), asbestos was referred to as *lithios amiantos*, meaning the undefiled rock retrieved from the fire. Herodotus referred to it as *linum vivum*, meaning the

Figure 2 Erionite as rock in hillside and cut into blocks for construction of a house in Capadocchia.

living cloth in reference to a funereal dress that would survive cremation of nobility.[14]

Idiosyncratic usage of asbestos prior to the industrial age

Asbestos allegedly was used as a coating for the feet of individuals who were undergoing trial by fire during the middle ages. Charlemagne (approximately 800 AD) allegedly had a noncombustible tablecloth made of asbestos. Marco Polo (approximately 1250 AD) allegedly had a similar cloth that could be purified by fire. Asbestos was used as a component of body armor of knights in France (approximately 1490). Benjamin Franklin (approximately 1725) had a purse made of asbestos to safeguard his money in case of conflagration.[14]

Commercial usage of asbestos

Asbestos came into its own as a commercial product during the industrial revolution. Its thermal insulating properties have been used to insulate boilers and pipes carrying both hot and cold liquids in factories, refineries, power plants, ships, and both private and public buildings. Its light weight has come into play when using large amounts in transportation. Its ability to be woven into fabrics accounts for its use in various fire-proofing products and its use in friction products, such as brakes and clutches, and its use in gaskets. Its texture permits its use as a component of electrical cables, cement, shingles, roofing, and other construction materials.

The commonly known occupations associated with exposure to asbestos and development of disease include shipyard workers, merchant seamen working in ships with asbestos, military personnel aboard ships laden with asbestos, boiler makers, plumbers and pipe fitters, brick layers and masons working around asbestos block, carpenters working with and around asbestos products, petrochemical workers working around asbestos-containing products, power plant workers working around boilers, railroad workers, and workers in the steel and refinery and rubber industries. Occupations where workers have been exposed in various manners but are not as widely appreciated include custodial and maintenance workers, laborers doing clean-up, electricians working around cable insulated with asbestos, welders, mechanics, decorators, jewelers, bakers, carpenters, painters, plasterers and dry wall workers, as well as school teachers and managers and clerks coming into contact with asbestos in their place of employment.

Five acts in the history of asbestos disease

Like a Shakespearean tragedy, the story of asbestos disease can be narrated in five acts (Table 1). Dr. Irving Selikoff (Figure 3) and colleagues summarized three waves of asbestos in a multidisciplinary monograph.[15] The first wave of cases, beginning at the end of the 19th century and concentrated in South Africa and Europe, were miners who provided the material for production of raw asbestos (Figure 4) and millers of asbestos products, including asbestos cloth and block. The second wave of cases were tradesmen who applied and removed asbestos-containing products, particularly including insulation (Figure 5). The third wave is exposure of bystanders to asbestos in place (Figure 6), including persons who are not aware that they are being exposed to asbestos, such as janitors and schoolteachers. A fourth act can be identified as persons exposed through family members by contaminated clothes brought into the home. These cases are commonly referred to as household or spousal exposure. A fifth act contains patients whose exposure is occult by virtue of the passage of time, failed memory, lack of knowledge by the patient as to the existence or significance of the asbestos with which he or she came into contact, suppression of information, or lack of appreciation of the degree of oncogenictiy of asbestos fibers. Removal of asbestos under controlled conditions is

Table 1 Five acts in the histroy of asbestos disease

Act 1. Miners and millers, handling and refining the product.
Act 2. Tradesmen, applying and removing the product.
Act 3. Bystanders to asbestos in place, usually not aware of it.
Act 4. Family members, exposed at home to contaminated clothes.
Act 5. Occult exposure, no longer possible to identify because of lack of notation in the medical record, the passage of time and failing of memory, the lack of knowledge of the patient or coworkers exposed, the death of the patient or coworkers, or the suppression of information.

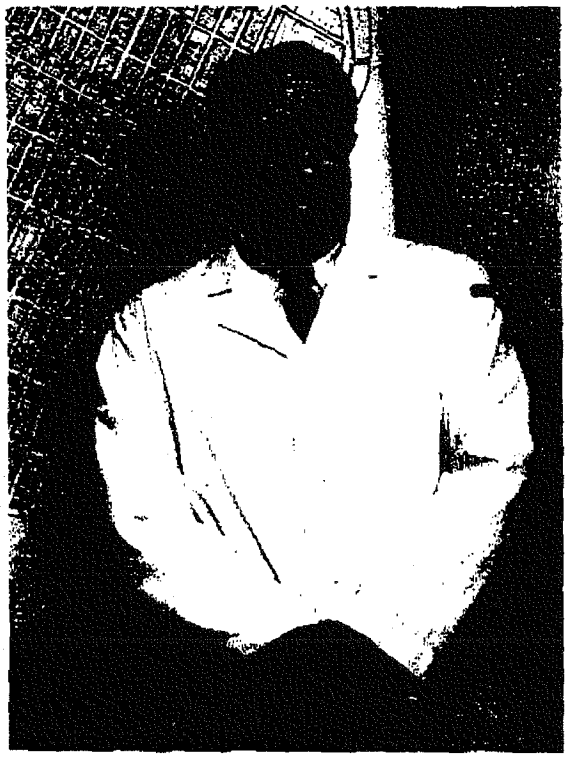

Figure 3 Dr. Irving Selikoff, whose work is a foundation for the study of asbestos-related diseases.

important in remediation of the effects of the second and third waves (Figure 7).

The recognition of DMM by pathologists

Isolated cases of pleural tumors were recorded in the pathologic literature in the last two decades of the 19th century and the first four decades of the 20th century. Books and review articles dealing with tumors of the lung tended to ignore pleural tumors, a fact which suggests either that the authors were not aware of the literature, had not seen cases themselves, or did not believe that such cases differed from the carcinomas of the lung, which themselves were uncommon at the time.

Figure 5 Asbestos insulation around pipes, deteriorating.

The oldest citations

There are citations using the term mesothelioma from the 18th and 19th centuries, the earlier ones preceding Virchow's cellular theory of disease and the routine use of microscopy, so one cannot draw specific conclusions as to what the authors were describing. Many terms were used for pleural-based tumor. In particular, the term endothelioma was used to indicate a purported histogenesis from endothelial cells of pleural lymphatics or pleural capillaries.

The early German literature

The known field of pathology in the 1920s and 1930s was reviewed in 30 volumes of the *Handbuch der speziellen pathologischen Anatomie und Histologie*,[16] edited by F.

Figure 4 Asbestos wool, the raw product after milling and ready for fabrication into specialized products.

Figure 6 Asbestos for bystanders, having fallen onto the floor of a basement corridor.

Figure 7 Emblem of an asbestos removal unit in the United States Army, showing a bear with tools in one hand and flocks of asbestos in the other hand.

Henke of Breslau, Germany and G. Lubarsch of Berlin, Germany (Figure 8). The majority of the findings were based on the autopsy. The contributing authors, although primarily German, exhaustively reviewed English, French, Spanish, Italian, Russian, and other literature. Three volumes of the Handbuch, encompassing 2268 pages, were devoted to the lung and pleura. Ninety-eight pages were devoted to lung tumors, including 1100 references, a review of 1888 cases in the literature, and a subclassification of the common carcinomas, which is essentially the same subclassification that is used today. Four pages each were devoted to anomalies of the pleura and to tumors of the pleura. The author listed the many names used for supposedly primary pleural tumors. These included endothelioma, endothelial carcinoma, endothelial sarcoma, lymphangioendothelioma, perithelioma, mesothelioma, and malignant mesothelial tumor. The frequency with which these tumors metastasized to lymph nodes, brain, liver, and adrenal glands were recorded. Primary fibrosarcomas of the pleura were illustrated (Figure 9). The Handbuch quoted authors who believed that pleural tumors were specific and quoted authors who were convinced that primary pleural tumors did not exist. This discussion occurred at a time when the biphasic nature of mesothelial cells, capable of displaying both epithelioid and mesenchymal features, had already been established by experimental studies and embryological principles.

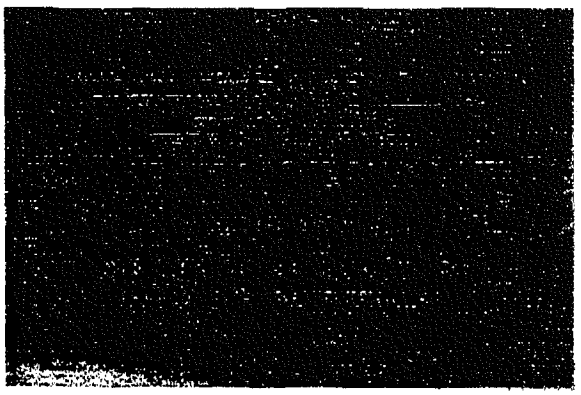

Figure 8 The encyclopedic *Handbuch* edited by Henke and Lubarsch, with one volume devoted to lung tumors, published in 1931.

Figure 9 Fibrosarcoma of the pleura illustrated in the Henke and Lubarsch *Handbuch*, where the distinction is made between this tumor and the absence of any well documented mesothelioma.

In 1931, after considering the possibilities, the Handbuch concluded as follows: "In der überwiegenden Mehrzahl der Fälle ist zweifellos der sog. primäre Pleuratumor eine Metastase oder eine Ausdehnung eines primaeren Lungenkrebses auf die Pleura. Ich bin fest überzeugt, daβ, wenigstens was die Pleura betrifft, keine Diagnose auf primären Krebs, Mesotheliom, oder Endotheliom, sei es ausgehend von den Lymphgefäßendothelien, sei es von den serösen Deckzellen, irgendwie logisch begründet werden kann." (In the overwhelming number of cases doubtless the so-called primary pleural tumor is a metastasis or an extension of a primary lung cancer to the pleura. I am firmly convinced that, at least with regard to the pleura, no diagnosis of primary carcinoma, mesothelioma or endothelioma of the pleura can be logically justified, whether originating from lymph vessel endothelium or from serosal surface cells.)

In 1932, Fischer-Wasels[17] in Frankfurt am Main, Germany concluded that authentic benign or malignant tumors that consist of serosal cells or derived form serosal cells were as yet unknown.

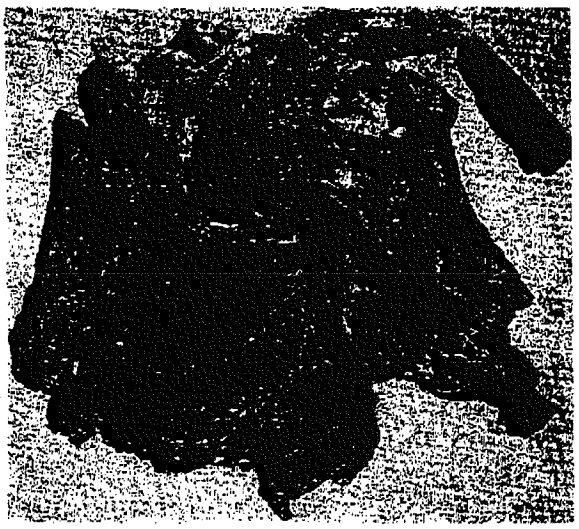

Figure 10 First case of DMM diagnosed at the Massachusetts General Hospital, illustrated in a clinicopathological conference in 1947 with neoplastic involvement of visceral pericardium.

In 1943, Wedler[18] in Heidelberg, Germany reported on a series of autopsies of 30 asbestos workers. One patient was 18 years old and had no asbestosis. The remainder were 31 years or older and had some degree of asbestosis. Four of these patients died of bronchogenic carcinoma, which was distinguished from 2 who died of pleural malignancy. He described the absence of a pulmonary mass in these 2 cases and connected the work with asbestos to all of the cases. He also referred to descriptions of individual cases of "pseudoalveolaeres mesotheliom" in the literature. Since he took pain to distinguish primary lung tumors from primary pleural tumors, it is fair to conclude that he was describing malignant mesotheliomas in asbestos workers.

In 1948, Walther[19] from Zurich, Switzerland authored a 560-page treatise on the metastases of cancer (Krebsmetastasen) and devoted 10 pages to spread of malignant tumors in serosal cavities. He reviewed 422 cases of malignant tumors involving the pleural cavity with 47 different origins and 505 cases of malignant tumors involving the peritoneal cavity with 31 different origins. There is no mention of mesothelioma.

The early English-language literature

The first case of DMM at the Massachusetts General Hospital was diagnosed in 1946, when a mesothelioma of the pleura was published as a Case Record of the Massachusetts General Hospital.[20] The patient was a 37-year-old Swedish asbestos worker. The chest radiographs showed a large intrathoracic mass which was outside the lung. The clinical diagnosis was either carcinoma of the lung or mediastinal teratoma. Autopsy showed that the left lung was completely encased and the pericardium thickened (Figure 10) by a hard fibrous tumor, composed histologically of cells that in areas were cuboidal and formed papillae (Figure 11). The anatomical diagnosis was mesothelioma of the pleura and pericardium. Dr. Benjamin Castleman (Figure 12), the pathologist discussing the case, commented that "a number of papers have been written to the effect that there is no such tumor as mesothelioma of the pleura, that the cells lining the pleura do not form tumors and that these tumors really arise from a small focus in the lung. We have held a similar opinion for a long time. This is perhaps the first case in which we believed that there was actually such a tumor. It certainly fits in with most of the cases of mesothelioma of the pleura that have been reported." The clinician who discussed the case rejoined that "I do not consider that it is fair to have given me a case with a diagnosis against which you, as pathologists, have been talking for twenty years. I could never make Dr. Mallory accept a diagnosis of mesothelioma of the pleura." Dr. Castleman thereby became the first pathologist to illustrate the details of a DMM in a person in the United States who worked with asbestos. Another early report of DMM appeared as a clinicopathologic conference at Barnes Hospital, St. Louis, Missouri in 1949.[21]

Dr. R.A. Willis, a renowned pathologist who served as consultant pathologist to the Imperial Cancer Research Fund, London, England, authored the widely used textbook *Pathology of Tumors*. The book went through several editions. In the third edition published in 1960 (Figure 13), Dr. Willis made the following statement: "Thus, to the present day, the accounts of supposedly primary serosal tumors continue to be descriptively inadequate and insufficiently

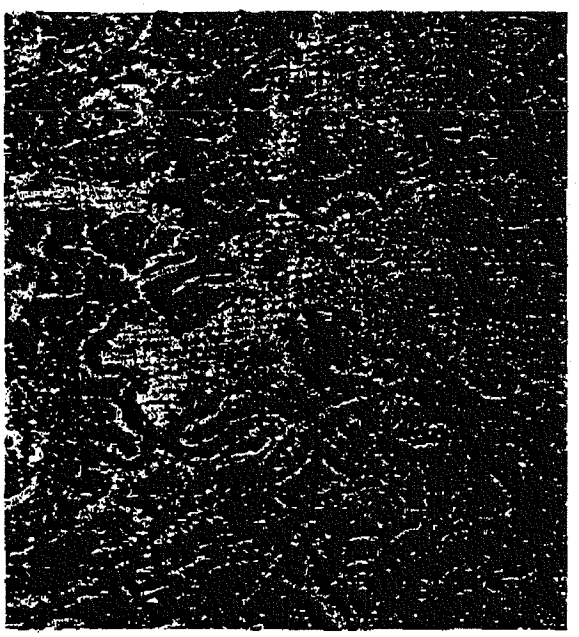

Figure 11 Papillary fronds of cuboidal cells of a DMM, as published in the clinicopathological conference.

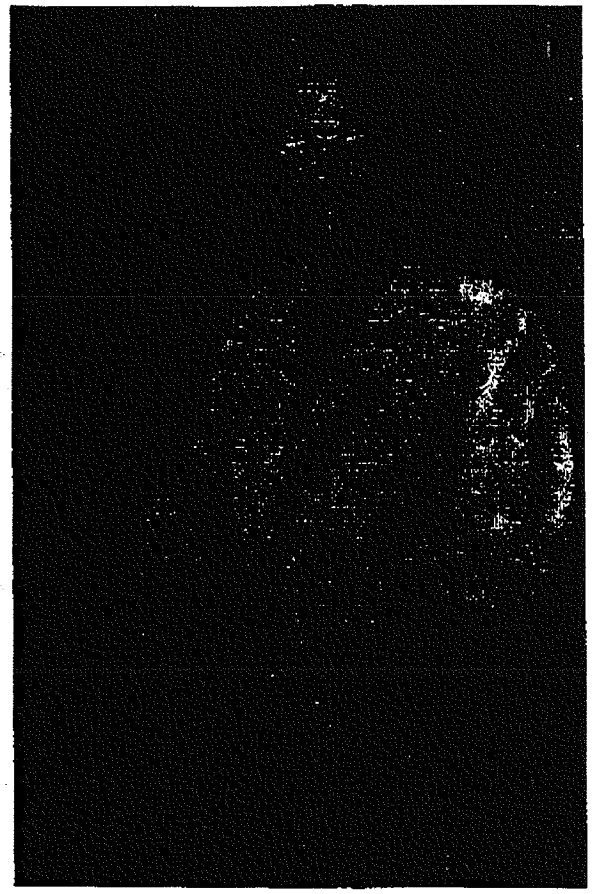

Figure 12 Dr. Benjamin Castleman, the pathologist who discussed this initial case.

critical, displaying a lack of awareness of the possible behavior of small undetected carcinomas. The only tumor which I myself have ever ventured to suggest might be a primary one of a serous membrane was in a case reported in 1938. This was a polypoid growth inside a large surgically removed hydrocele sac; subsequent necropsy showed this to have been a metastasis from a small unsuspected bronchial carcinoma."[22]

Subsequent recognition of DMM and its causation by asbestos

Two cases of DMM were reported in 1958 in asbestos workers in Holland.[23] Thirty-three cases of DMM were reported in 1960 from South Africa.[24] The patients worked in or around asbestos mines or were family members of the miners. Selikoff, in 1965, reported on the development of DMM and follow-up of insulation workers in New Jersey.[25] Hillerdahl's review of 4710 published cases of DMM and discussion of the relationship to asbestos appeared in 1983.[26] Mancuso reported in 1991 on DMM of epidemic proportions in railroad workers.[27]

Subsequent reports of DMM and their relation to asbestos continue to appear in increasing numbers. Older reports describe other causes of DMM.[28] Erionite causes DMM.[29] Because therapeutic radiation induces other tumors, it is plausible that therapeutic radiation can induce DMM. There are familial cases of DMM, and individual susceptibility may play a part in an individual who is unlucky enough to develop DMM. However, other purported causes lack scientific basis based on molecular medicine and do not satisfy the nine criteria of causation as set forth by Bradford Hill.[30]

Was DMM simply confused with other tumors, or is it a new disease?

Diseases can change names over time, for a variety of reasons. A new disease must be distinguished from a disease with a new name or which acquires new criteria for diagnosis over time. However, not all diseases have existed throughout human history.[1] New diseases with distinctive clinical and pathologic features that have appeared in the last half of the 20th century include acquired immunodefi-

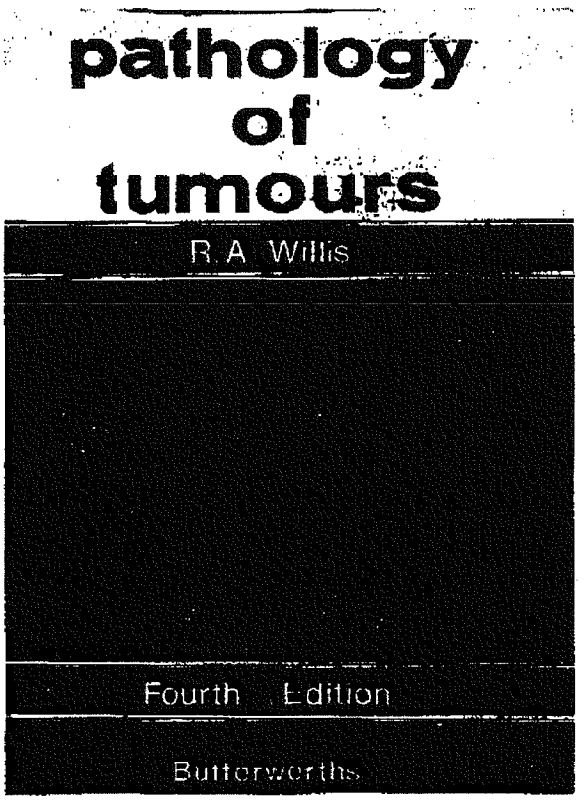

Figure 13 The widely used treatise *Pathology of Tumors* by Dr. Willis, third edition published in 1960, where malignant mesothelioma is not accepted as an entity.

Table 2 Mesothelial tumors other than diffuse malignant mesothelioma

Localized fibrous tumor of the pleura (fibrous mesothelioma)
Malignant form of localized fibrous tumor of the pleura (fibrosarcoma)
Papillary surface tumor of the ovary (mesothelioma of the female genital tract)
Adenomatoid tumor (mesothelial tumor of paratesticular tissue and other sites)
Mesothelioma of the atrioventricular node
Benign mesothelioma of the peritoneum (multicystic mesothelioma)
Well-differentiated papillary mesothelioma
Mesothelioma of infancy and childhood

ciency syndrome due to human immunodeficiency virus, clear-cell carcinoma of the vagina associated with administration of diethylstilbestrol during pregnancy, pulmonary arteriopathy due to administration of appetite suppressants in Switzerland in the 1960s and in the United States in the last decade, hepatic angiosarcoma due to administration of Thorotrast, lead toxicity due to the use of lead-based products, and amiodarone pneumonitis due to the administration of amiodarone. Other diseases disappear, at least in large populations. Examples are smallpox, diphtheria, scurvy, and rickets as well as disease due to administration of the above listed substances when those substances are removed from contact with patients.

Is DMM a new disease? Or has it acquired different or more technically sophisticated clinical and pathological criteria for diagnosis? And why should one care whether a disease is new or simply has a new name? The importance of the distinction derives from the following syllogism: a tumor is a disease; a disease has a cause; a new tumor has a new cause.[1]

Mesothelial tumors arise by definition from mesothelial cells. However, tumors other than diffuse malignant mesothelioma may have an origin from mesothelial cells or submesothelial fibroblasts. Other forms of mesotheliomas or tumors derived from serosal or subserosal cells include localized fibrous tumor of the pleura, fibrosarcomas of the pleura as the malignant counterpart of localized fibrous tumor, papillary surface tumors of the female pelvis, adenomatoid tumors, mesothelioma of the atrioventricular node of the heart, multicystic mesothelioma of the peritoneum, well differentiated papillary mesotheliomas of the peritoneum and rarely of the pleura, and mesotheliomas of infancy and childhood. These are listed in Table 2.

Clinical and pathological criteria differ. Some clinical criteria for the diagnosis of diffuse malignant mesothelioma of the pleura include a pleural-based tumor, pleural effusion, local invasion causing pain, weight loss, recurrence in a needle tract, and metastasis. The changing pathological criteria are given in Table 3. They have included, in succession historically, pleural based tumor, serosal spread, biphasic morphology, histochemistry, electron microscopy, and immunochemistry

Increasing awareness of a disease among pathologists increases the likelihood of considering, and then making, the diagnosis. Increasingly sophisticated tests for diagnosing diffuse malignant mesothelioma could either increase or decrease the number of times the diagnosis is made. Either possibility exists. Immunochemical criteria have made pathologists more confident in differentiating metastatic adenocarcinoma or sarcoma in the pleura from DMM, enabling pathologists to include or exclude DMM in instances when no other primary tumor is evident and when an autopsy has not been performed. By analogy with other areas of histopathology, the increasing experience gained by pathologists who have seen many cases of DMM can enable them to make diagnosis of DMM on the basis on slides stained with hematoxylin and eosin alone in many instances.

The inverse relationship between the prevalence of fibrous tumors of the pleura and DMM

An inference about the increasing incidence of DMM can be obtained from the relative number of reports of localized fibrous tumors of the pleura (fibrous mesothelioma) compared with DMM. Five cases of primary neoplasms of the pleura were studied at the Mount Sinai Hospital in New York City.[31] Three of the cases were large, circumscribed, pedunculated fibrous tumors of the visceral pleura which grew slowly and today would be termed localized fibrous tumors of the pleura. One case was a giant lipoma of the pleura. The fifth case was a diffuse pleural neoplasm which encased the lung and whose illustrated histologic pattern comports with that of a DMM. That the authors could accumulate four examples of the relatively uncommon localized pleural tumor and just one example of a DMM bespeaks the great rarity of DMM at the time. A similar

Table 3 Diffuse malignant mesothelioma: changing criteria for pathologic diagnosis

Tumor involves the pleura and no other tumor is identified.
Tumor spreads selectively upon the serosa.
Tumor invades into the interstitial layer of the pleura and subsequently deeper.
Histologic pattern may be biphasic (epithelioid and mesenchymal), reflecting the schizocoelomic embryology of the pleural cavity.
Histochemistry (historically periodic acid-Schiff after digestion with diastase and alcian blue after digestion with hyaluronidase).
Ultrastructure (epithelioid cells with long and thin and branching microvilli).
Immunochemistry (changing and enlarging panels of reagents).

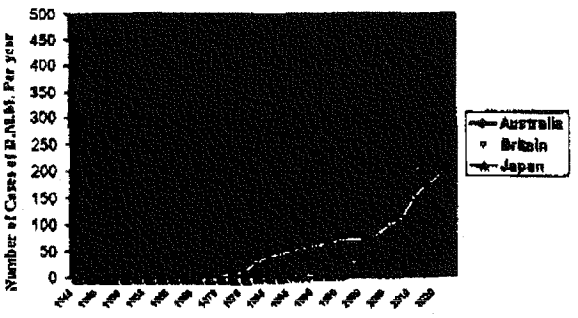

Figure 14 Composite graph of the rising incidence of DMM in three countries and, retroactively, the absence of the disease before 1960. Data taken from reference 34 (Great Britain), reference 35 (Australia), and reference 36 (Japan). Cases in Great Britain are males only.

report of a low ratio of localized fibrous tumors of the tumor to DMM in a series of four cases appeared in 1955.[32] Today, the ratio is reversed. DMM burgeons while localized fibrous tumors of the pleura are static in the authors' experience.

The increasing incidence of DMM into the 21st century

The statement is commonly made that DMM is a rare disease, and there was a time when that was true. However, thousands of new cases per year now[33] would not constitute a rare disease in the minds of many persons. In Great Britain,[34] Australia,[35] Japan,[36] and the United States,[37] there have been multifold increases in DMM over intervals as short as two decades with projected peaks between the years 2000 and 2025, depending on the country[38] and its respective use of asbestos.

A graphic display of the increasing incidence in three countries is given in Figure 14. All three lines on the graph extrapolate back in time to an effective incidence of zero before 1960, indicating that there are virtually no cases that should be called background. Because these trends mirror the usage of asbestos in these three countries with a delay defined by the latency of DMM, and with what we know about the carcinogenicity of asbestos, it follows that there are virtually no cases unrelated to asbestos, and to call selected cases "spontaneous" or "idiopathic" is a mistaken concept.

Discussion

The significance of a signal tumor is that it tells us that something new has happened. Among the explanations put forth that some mesotheliomas are not due to asbestos is the concept that there is a background level of disease that has been present throughout human history.[39] Did DMM afflict mankind before the commercial application of asbestos? Sporadic albeit undocumented cases probably occurred, but this article has shown that there was also sporadic use of asbestos before its commercial use in the industrial age. The uncertainty of the very existence of DMM into the 1940s and 1950s indicates its true rarity at that time. A more recent review of nonoccupational and low-dose exposures states that "There might exist a background level of mesothelioma occurring in the absence of exposure to asbestos, but there is no proof of this and this 'natural level' is probably lower than the 1 to 2 million/yr which has often been cited."[6]

Physicians can diagnose a disease only when they know that it exists, so one explanation for the rise in incidence is more widespread appreciation of the disease. However, most cases of DMM are suspected by macroscopic and routine microscopic techniques that have been available since the beginning of the 20th century. Modern pathology has proscribed additional requirements for the diagnosis of DMM, but a priori one cannot assume that these requirements have increased the likelihood of the diagnosis, because the requirements also can serve to exclude the diagnosis. The rarity of the disease cannot be attributed to failure in identification, because pathologists had discussed its possible existence and theoretical attributes and overall still did not identify it.

Building materials with asbestos are manufactured in about 100 countries. Chrysotile now accounts for approximately 95% of total usage of asbestos. Asbestos cement makes up 85% of current commercial use worldwide. Friction materials, floor tiles, gaskets, insulation board, and textiles are other major uses.[33] Occult sources of exposure to asbestos and unexpected release of asbestos[40] continue to come to public attention, and recent studies document the importance of very low exposures to asbestos and the importance of each exposure as it contributes to the cumulative exposure that ultimately causes DMM in a given individual. DMM looms as a large financial burden for patients and for society.[40] The duration of the rise and time of reversal will depend on the quantity and manner of usage of asbestos and the public health efforts to prevent exposure to asbestos already in place.

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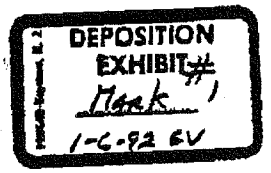

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TEACHING PORTFOLIO

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A. SELF REPORT OF LOCAL CONTRIBUTIONS: HMS AND HOSPITAL TEACHING EFFORT

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1974-76 HMS II, Skin Pathophysiology, laboratory instructor.

1977-79 HMS II, Lung Pathophysiology, laboratory instructor.

1980-1991 HMS-MIT Division of Health Sciences and Technology, second (and occasionally first) year Lung Pathophysiology, lecturer and laboratory instructor, sole pathologist in course, four to eight hours of lectures and demonstrations spaced over two months per year, approximately 40 students per year. Special accomplishment: Socratic method of teaching gross pathology, taxing students' powers of observation using fresh and fixed specimens from operations and autopsies performed during the days prior to lecture. Question and answer format used during entire lecture for continuous assessment of students' comprehesion of histopathology, since the ability of most of the students to recognize normal histology has been marginal. Emphasis on anatomic subdivisions of the lung applied to mechanics of ventilation, in an effort to utilize strong mathematical background of most students. Highest rating of instructors in pulmonary pathophysiology section for 1991.

2. MEDICINE, SURGERY, PEDIATRICS AND RADIOLOGY RESIENTS (Massachusetts General Hospital)

1974-1991 Monthly clinical rounds, presenting pathologic findings of specific patient and correlating with clinical or radiographic findings, discussing general principles of pathology and of the disease in question. Review of cases with individual residents as requested.

3. PATHOLOGY RESIDENTS (Massachusetts General Hospital)

1974-1991 Daily teaching of general surgical and autopsy pathology while working together on current patients, principally using a double-headed microscope Reviewing all complicated lung biopsies with individual resident responsible for the case, done on a daily basis. Biweekly didactic conference on pulmonary pathology, one hour in length, using difficult cases referred to me as consultations from outside hospitals, after the residents have had an opportunity to review the microscopic slides as well as clinical story and x-rays.

4. INVITED PRESENTATIONS (RECENT) WITHIN THE HOSPITAL AND MEDICAL SCHOOL AND OTHER LOCAL MEDICAL SCHOOLS

1985 Pathology Grand Rounds at Tufts-New England Medical Center, "Diagnostic Dilemmas in Lung Neoplasia."

1986 Pathology Grand Rounds at Brigham and Women's Hospital, "Systemic Approach to the Lung Biopsy."

1986 Infectious Disease Grand Rounds at Massachusetts General Hospital, "Histology of Opportunistic Infections in the Lung."

1987 Pathology Grand Rounds at University of Massachusetts Medical Center, "Usual interstitial pneumonitis."

1988 Pathology Grand Rounds at Massachusetts General Hospital, "Medical resources in Grenada, West Indies."

1988 Clinicopathologic Conference at Boston University Hospital, "Wegener's granulomatosis."

1989 Pathology Grand Rounds at Mallory Institute of Pathology, "Usual interstitial pneumonitis: Pathogenesis, diagnosis and implications of the diagnosis."

1989 Immunology Grand Rounds at Massacusetts General Hospital, "Immunologic Lung Disease."

1990 Boston Inter-Hospital Chest Clinic, "Quarterly Rounds of Interesting Cases."

1990 Boston Combined Hospitals Pediatric Pulmonary Conference, "Bimonthly review of difficult cases."

1991 Pathology Grand Rounds at Beth Israel Hospital, "Artefact and reality in the histologic diagnosis of interstitial lung disease.

5. POST-GRADUATE COURSES (Harvard Department of Continuing Education)

1977- Faculty, Harvard-Massachusetts General Hospital Thoracic Surgery Post-Graduate Course, Current Concepts in Thoracic Surgery.

1978- Faculty, Harvard-Massachusetts General Hospital Pathology Post-Graduate Course, Current Concepts in Surgical Pathology.

1978-1979 Faculty, Harvard-Massachusetts General Hospital Internal Medicine Post-Graduate Course, Current Concepts in Internal Medicine.

1978- Faculty, Harvard-Massachusetts General Hospital Pulmonary Medicine Post-Graduate Course, Current Concepts in Pulmonary Medicine.

1989- Faculty, Harvard-Massachusetts General Hospital Thoracic Surgery Post-Graduate Course, Surgery of the Upper Airways.

1985-1990 Director, Harvard Medical School Post-Graduate Course, Current Concepts in Pulmonary Pathology.

1991- Codirector, Harvard-Massachusetts General Hospital Pathology Post-Graduate Course, Current Concepts in Surgical Pathology.

1992- Faculty, Harvard-Massachusetts General Hospital Post-Graduate Course, Current Concepts in Infectious Disease.

I founded the week long course "Current Concepts in Pulmonary Pathology" in 1985 singlehandedly and have been the sole director of the course since its inception. It is the only, current, annual, in-depth course on pulmonary pathology in the world. The course has had a steady enrollment of 60-70. attendees, who are pathologists or chest physicians. in universities or in private practice. An intrinsic part of the course, which draws many of the students, is the unique laboratory. This consists currently of 3000 consultation cases referred to me, consisting of microscopic slides together with specimen photographs, clinical records, radiographs, my report, and follow-up. Each case is a self-contained instructional unit.

6. FELLOWS (FULL TIME)

Jose R. Ramirez, M.D., Department of Pathology, University of Barcelona, Barcelona, Spain (1984-1985)

Osamu Matsubara, M.D., Department of Pathology, Tokyo Medical and Dental College, Tokyo, Japan (1987-1988)

Nelia S. Tan-Liu, M.D., Department of Pathology, University of the Philippines, Manila, Philippines (1987-1988)

Yukio Nakatani, M.D., Department of Pathology, Yokohama City University, Yokohama, Japan (1988-1989)

Toyohara Yokoi, M.D., Department of Pathology, Nagoya University School of Medicine, Nagoya, Japan (1989-1990)

Dong-Hwan Shin, M.D., Department of Pathology, Yonsei University College of Medicine, Seoul, Korea (1989-1991)

7. ADVISING RESPONSIBILITY

Advisor to one new resident per year on average (since 1980) for the duration of that resident's tenture at the Massachusetts General Hospital.

B. SELF REPORT OF NATIONAL AND INTERNATIONAL CONTRIBUTIONS.

1. INVITED PRESENTATIONS, VISITING PROFESSORSHIPS AND LECTURESHIPS

1974 United States Military in Europe Medical-Surgical Congress, Garmisch, Germany

1975 Valley Medical Center, Fresno, California

1977, 1980, 1983, 1985 New England Society of Pathologists, Boston, Massachusetts

1977 New England Society of Pathology Residents, Boston, Massachusetts

1978 United States Naval Submarine Medical Center, Groton, Connecticut

1979 Memphis Society of Pathologists, Memphis, Tennessee

1979 Baptist Hospital and Le Bonheur Childrens Hospital, University of Tennessee Medical School, Memphis, Tennessee

1979, 1982, 1984, 1985 Brooke Army Medical Center, San Antonio, Texas

1980 Greeneville Society of Pathologists, Greeneville, South Carolina

1980 Department of Pathology, University of South Carolina Medical School, Columbia, South Carolina

1981 William Beaumont Army Medical Center, El Paso, Texas

1981 American Osteopathic College of Pathologists, Boston, Massachusetts

1982, 1983 Booth Memorial Medical Center, New York University School of Medicine, New York, New York

1982 Manitoba Pathology Society, Winnipeg, Manitoba

1982 Guest faculty, 20th Annual Course, Pathology of the Lung, Department of Pathology, University of California San Diego School of Medicine, San Diego, California

1983, 1984 General Carl W. Tempel Pulmonary-Allergy Symposium, Fitzsimmons Army Medical Center, Denver, Colorado

1983 Department of Civil Engineering, Tufts University, Medford, Massachusetts

1983 Department of Pathology, Rutgers University, New Brunswick, New Jersey

1984 Letterman Army Medical Center, San Francisco, California

1984, 1985 California Society of Pathologists: San Francisco, California; Newport Beach, California

1985 Boca Raton Annual Cancer Symposium, Boca Raton, Florida

1985 Naples Community Hospital, Naples, Florida

1985 Department of Pathology, New England Medical Center, Tufts University Medical School, Boston, Massachusetts

1985 Malden Hospital, Malden, Massachusetts

1986 Department of Pathology, Yale-New Haven Medical Center, New Haven, Connecticut

1986 Chicago Pathology Society, Chicago, Illinois

1986 Walter Reed Army Medical Center, Washington, D.C.

1986 Department of Pathology, St. James' Hospital, Dublin, Ireland

1986 Associacion de Anatomia Patologica de Cataluna y Baleares, Barcelona, Spain

At the request of the Univesity of Barcelona, I gave a two day seminar on pulmonary pathology This constituted the keynote address and major business of the annual meeting of this spanish society of pathology.

1986 Pathology Society of Philadelphia, Philadelphia, Pennsylvania

1987 Department of Pathology, University of Massachusetts Medical School, Worcester, Massachusetts

1987 25 th Schleifstein Conference, New York State Association of Public Health Laboratories, Albany, New York

I was the keynote speaker and seminar director for this annual honorary conference.

1987 Ministry of Health, Republic of Singapore, Singapore

At the request of the Government of Singapore, I delivered a two week course in pulmonary pathology of the lung, given several hours per day to the medical students and chest physicians and pathologists at the National University.

1987 Department of Pathology, Tokyo Medical and Dental University, Tokyo, Japan

1987 Department of Pathology, Fukuoka University Medical School, Fukuoka, Japan

At the request of two universities in Japan (Kurume, Toyko Medial and Dental), I gave a series of lectures covering the field of lung pathology, over two and three days at each institution, respectively

1987 Department of Pathology, Marshall University School of Medicine, Huntington, West Virginia

1987 River Cities Pathology Society, Huntington, West Virginia

1988 Department of Pathology, University of Nebraska Medical Center, Omaha, Nebraska

1988 Nebraska Association of Pathologists, Omaha, Nebraska

1988 Project HOPE, General Hospital, St. George's, Grenada, West Indies

1988 Oregon Thoracic Association, Hood River, Oregon

1989 Florida Society of Pathologists, Orlando, Florida

1989 Department of Pathology, New York Medical College, New York, New York

1989, 1990 Collegium Ramazzini; Ottawa, Ontario; New York, New York

I served as panel foreman during a three day conclave on "Disease potential of different asbestos fiber varieties" This conclave was sponsored by Collegium Ramizini, an honorary society of physicians interested in occupational medicine. I served on the organizing committee of the same society for a conclave on the effects of low-level exposure to asbestos, held in New York City in 1990.

1989 Department of Medicine, University Hospital, Boston University School of Medicine, Boston, Massachusetts

1989 U.S. Army Medical Research Institute of Chemical Defenses, Edgewood Arsenal, Aberdeen Proving Ground, Maryland

1989 Connecticut Society of Pathologists, Hartford, Connecticut

1989 St. Francis Hospital and Medical Center, Hartford, Connecticut

1989 Mallory Institute of Pathology, Boston University School of Medicine, Boston, Massachusetts

1990 Salem Hospital, Salem, Massachusetts

1991 Department of Pathology, University of Vermont, Burlington, Vermont

1991 Annual Sniffen Honorary Lecturer, Worcester Memorial Hospital, Worcester, Massassachusetts.

1992 XIX International Congress of the International Academy of Pathology & 10th World Congress of Academic and Environmental Pathology, Madrid, Spain.

1992 Sociedade Brasileira de Pneumologia e Tisiologia, Brasilia, Brasil.

The fifty-four items listed as "Visiting lecturer" in my Curriculum Vitae all represent invited presentations, where I was either the only guest lecturer or one of a few. Travel expenses, if any, and honorarium, if any, were all paid by host institution. Twelve host institutions or societies or universities were in New England, thirty-two in other parts of the United States, eight occurred outside the United States, and seven were at several different installations of the United States Army across the country. Several of the items incorporate multiple return visits. Duration of lectures or seminars or courses was one hour to two weeks. Major seminars, lasting three to eight hours with extensive preparation of slide sets and syllabi of 10 to 40 pages, occurred in Memphis, TN 9 L979); Greeneville, S.C. (1980); Winnipeg, Manitoba (1982); San Diego, CA (1982); San Francisco and Newport, CA (1984 and 1985); Boca Raton, FL (1985); Chicago, IL (1986); Philadelphia, PA (1986); Albany, NY (1987); Huntington, WV (1987); Omaha, Nebraska (1988); Portland, Oregon (1988); New York, NY (1989); Hartford, CT (1989).

2. PROFESSIONAL AND EDUCATIONAL LEADERSHIP ROLES

1978-1982 Faculty, International Academy of Pathology, three hour course "Skin Biopsy in Emergency Diagnosis" given together with Martin C. Mihm, M.D. on an annual basis.

1983-1986 Faculty, American College of Chest Physicians, three hours of lectures of "Pathology of Pulmonary Neoplasms," on an annual basis.

1985- Faculty, American Society of Clinical Pathologists, three hour seminar on "Biopsy Diagnosis of Pulmonary Tumors," given approximately on a biannual basis.

1985, 1987, 1989 Panelist, Evening Specialty Conference, "Pulmonary Pathology," International Academy of Pathology, annual meeting.

1989,-1993 Moderator, Evening Specialty Conference, "Pulmonary Pathology," International Academy of Pathology, annual meeting.

As director of the panel, I chose panel members, suggest topics for discussion, organize syllabus, direct discussion, and implement suggestions on evaluation forms for the following year.

TEACHING AWARDS

I have been awarded the specialty identification MOS 61 V 9A/9B/9R/9Z (professor) by the United States Army for service to Army medical education programs in teaching hospitals across the county. I have done this teaching as part of my role as a colonel in the medical corps of the United States Army Reserve.

Military Appointments:

Security clearance: Top Secret

Primary Specialty Skill Identifier: 6l V 9A/9B/9R/8Z

Secondary Specialty Skill Identifier: 6l U 9B/8B

1972 - 1974	Major, U. S. Army, European Area Reference Laboratory, Landstuhl, Germany
1975 - 1977	Major, U.S. Army Reserve, 323rd Medical Laboratory, Boston, Massachusetts
1978 - 1981	Lieutenant Colonel, U.S.Army Reserve, 323rd Medical Laboratory, Hanscom Air Force Base, Massachusetts
1982	Graduate, Command and General Staff Officer Course, Medical Division, Fort Sam Houston, San Antonio, Texas
1983 - 1985	Colonel, U.S. Army Reserve, 323rd Medical Laboratory, Hanscom Air Force Base, Massachusetts
1986 -	Colonel, U. S. Army Reserve, 373rd General Hospital, Boston, Massachusetts
1991	Graduate, The Armed Forces Combat Casualty Care Course, Uniformed Services University of the Health Sciences, Fort Sam Houston, San Antonio, Texas